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Association between body size phenotype and sleep duration: Korean National Health and Nutrition Examination Survey V (KNHANES V)

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ABSTRACT

Objective. Recent studies reported the presence of unique subsets of body size phenotypes that are more susceptible or more resistant to the development of obesity-associated metabolic disorders, although the underlying mechanism is not yet fully elucidated. We investigated the association between body size phenotypes and sleep duration after adjusting potential confounding factors.

Materials and methods. We analyzed data from the Korean National Health and Nutrition Examination Survey V (KNHANES V), a nation-wide, population-based health survey including 9077 Korean adults. The average amount of sleep per night was categorized as: ≤6, 7, 8, and ≥9 h. Body size phenotypes were classified based on body mass index (BMI) and presence of metabolic syndrome; metabolically healthy and normal weight (MHNW), metabolically abnormal but normal weight (MANW), metabolically healthy but obese (MHO), and metabolically abnormal obese (MAO).

Results. According to sleep duration, there were significant differences in age, gender, BMI, waist circumference, and blood pressure (all $P < 0.05$). Multivariate analysis showed that obese groups (MHO and MAO) had significantly shorter sleep durations than non-obese groups (MHNW and MANW) (6.78 ± 0.04 vs. 6.93 ± 0.03, $P < 0.001$). Sleep duration was significantly different according to body size phenotype, irrespective of confounding factors, such as age, gender, smoking status, alcohol consumption, physical activity, income, and education (MHO; 6.73 ± 0.05, MAO; 6.82 ± 0.05, MHNW; 6.94 ± 0.04, and MANW; 6.91 ± 0.05; $P < 0.001$).

Conclusion. Sleep duration is independently associated with body size phenotype after adjusting for confounding factors in the Korean population.

Abbreviations: KNHANES V, Korean National Health and Nutrition Examination Survey V; BMI, body mass index; MHNW, metabolically healthy and normal weight; MANW, metabolically abnormal but normal weight; MHO, metabolically healthy but obese; MAO, metabolically abnormal obese; WC, waist circumference; FPG, fasting plasma glucose; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglycerides; HDL, high density lipoprotein cholesterol; HbA1c, hemoglobin A1c.

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1. Introduction

In parallel with an increasing prevalence of obesity, a reduction in sleep duration due to the demands of modern society has become more prominent in recent years. The self-reported sleep duration of Americans has decreased by 1.5–2 h over the last 40 years [1]. In 2009, the National Sleep Foundation reported that American adults sleep an average of 6 h and 40 min on weekdays [2]. Sleep duration and quality can significantly affect appetite, food intake, and energy balance, which may result in an increase in obesity [2]. Therefore, much attention has been paid to the association between sleep duration and obesity and obesity-related disorders.

Epidemiological and experimental studies have provided evidence that short sleep duration induces metabolic alterations and contributes to the development of obesity, insulin resistance, and type 2 diabetes [3,4]. A meta-analysis including 634,511 participants demonstrated that a pooled odds ratio for short duration of sleep and obesity was 1.55 (1.43 to 1.68; $P < 0.001$) [5]. This study showed that a reduction of 1 h of sleep per day was associated with a 0.35 kg/m^2 increase in body mass index (BMI) [5]. Cappuccio et al. reported that quantity and quality of sleep consistently predicted the risk of the development of type 2 diabetes in their meta-analysis including 107,756 participants [6]. Furthermore, another recent meta-analysis demonstrated that both short and long sleep durations are associated with an increased risk of prevalent hypertension [7]. We previously reported that both short and long sleep durations were related to an increased risk of metabolic syndrome and its components using representative national survey data including 4222 Korean men and women [8]. Patel et al. reported that mortality risk was lowest among women reporting a sleep duration of 7 h and that sleep times of less than 6 h or more than 7 h remained associated with an increased risk of death after adjusting for various confounding factors [9]. Therefore, short and/or long sleep duration may lead to increased mortality as a result of adverse effects on obesity, type 2 diabetes, and metabolic syndrome.

Recently, accumulating evidence has been found to support the idea that not all obese subjects are at similarly increased cardiometabolic risk and that patients with the metabolically healthy but obese (MHO) phenotype are protected from obesity-induced metabolic disorders and consequences. In contrast, patients with the metabolically abnormal but normal weight (MANW) phenotype, despite having a normal body mass index (BMI), exhibit greater visceral fat, atherogenic lipid profiles, and increased blood pressure and glucose levels, which ultimately contribute to the increased risk of cardiovascular disease (CVD) and mortality. In the South-West Seoul (SWS) study, we observed that elderly Koreans with the MANW phenotype showed a remarkably higher CVD mortality than MHO subjects, even after adjusting for other confounding factors [10]. Therefore, identification of underlying factors associated with MHO and MANW may provide more effective prevention and intervention strategies for obesity-related cardiometabolic consequences. We recently reported that low muscle mass (sarcopenia) is associated with different metabolic consequences according to body size phenotype in the Korean Sarcopenic Obesity Study (KSOS) [11]. However, although sleep duration has been reported to be closely associated with obesity and metabolic syndrome, to the best of our knowledge there have been no previous studies performed to evaluate the relationship between body size phenotype and sleep duration.

In the present study, we hypothesized that sleep duration may be associated with body size phenotype because it is known to association with body weight and metabolic syndrome. Therefore, we examined the association between sleep duration and body size phenotypes, including MANW and MHO, after adjusting for potential confounding factors using data from the Korean National Health and Nutrition Examination Survey V (KNHANES V), a nation-wide, population-based, cross-sectional health survey.

2. Subjects and methods

2.1. *Study population*

The Korean National Health and Nutrition Examination Survey V (KNHANES V) was performed by the Korean Ministry of Health and Welfare in 2010–2011. All study subjects underwent a health examination and finished a questionnaire, which involved demographic information, lifestyle behavior (exercise, smoking, and alcohol), education status, medical history, medication history, and sleep duration. Among a total of 23,524 participants over 20 years of age who finished the questionnaire, we excluded those without fasting time data (n = 1057), those who had a BMI less than 18 kg/m^2 (n = 286), those who had a chronic disease other than hypertension and diabetes, such as coronary heart disease, stroke, angina, thyroid disease, chronic liver or kidney disease, and cancers etc. (n = 12,517), and those who had missing data regarding sleep duration, BMI, and metabolic syndrome component (n = 587). Finally, 9077 Korean men and women were analyzed in the present study.

Sleep duration was assessed by the self-reported questionnaire. The categories were classified into four groups upon their sleep duration: less than 6 h/night, 6–6.9 h/night, 7–7.9 h/night, 8–8.9 h/night, and more than 9 h/night. Cigarette smoking was classified as none, ex-smoker (defined as a patient having smoked at least 5 packs throughout their entire life but did not currently smoke cigarettes), and current smokers. Alcohol drinking was categorized as follows: none, ex-drinker (defined as at least once per month for at least a year but who currently did not drink alcohol), and current drinker. Level of physical activity was classified into three groups: none, 1–4 times for at least 30 min per week, and 5 times for at least 30 min per week.

2.2. *Anthropometric and laboratory measurements*

Body weight and height were measured with subjects while they wore light clothing and no shoes. BMI was computed as weight (in kg) divided by height (in m^2). Waist circumference was measured from the narrowest point between the lower rib margin and the iliac crest. Blood pressure was taken by an average of two systolic and diastolic pressures which were measured with the subjects in a sitting position after at least

10 min of rest and with a 5 min interval. Fasting plasma sample was selected in the morning following a fast of at least an 8 h. Blood samples were centrifuged, refrigerated at the examination site, and transferred in ice boxes to a central laboratory in Seoul on the same day. Plasma glucose, total cholesterol, triglyceride, and HDL cholesterol were measured using an auto-analyzer (Hitachi Automatic analyzer 7600, Tokyo, Japan).

2.3. Definition of metabolic syndrome

To define metabolic syndrome, we used the harmonized criteria for clinical diagnosis of the metabolic syndrome recommended by a joint scientific statement [12]. Metabolic syndrome was defined as follows: (1) central obesity; (2) fasting triglyceride ≥1.7 mmol/l; (3) low high-density lipoprotein cholesterol (HDL-C) <1.0 mmol/l in males and <1.3 mmol/L in females; (4) hypertension with systolic/diastolic blood pressure ≥130/85 mmHg or antihypertensive drug use; (5) fasting plasma glucose ≥5.6 mmol/l or antihyperglycemic agent use. Waist circumference was considered to indicate central obesity at ≥90 cm in men and ≥80 cm in women, based on the World Health Organization Asia-Pacific criteria [13].

2.4. Definition of body size phenotypes

Four body size phenotypes were classified in the current analyses using combined consideration of BMI values, diabetes status, and the metabolic syndrome components. The present study used the obesity criteria of Korean Society for the Study of Obesity (KSSO) and WHO Asia-Pacific perspective, which define obesity as BMI ≥25 kg/m^2 in Asian population [14]. Normal weight without metabolic syndrome component: 18 kg/m^2 ≤ BMI <25 kg/m^2 and ≤2 metabolic syndrome components, but no diabetes [metabolically healthy and normal weight (MHNW)]; 18 kg/m^2 ≤ BMI <25 kg/m^2 and ≥3 metabolic syndrome components or diabetes [metabolically abnormal but normal weight (MANW)]; overweight/obese without metabolic syndrome components; BMI ≥25 kg/m^2 and ≤2 metabolic syndrome components, but no diabetes, and overweight/obese with metabolic syndrome components [metabolically healthy but obese (MHO)]: BMI ≥25 kg/m^2 and ≥3 metabolic syndrome components or diabetes [metabolically abnormal obese (MAO)] [15].

2.5. Statistical analysis

All the statistical analyses were performed by a professional statistician, one of our authors (J.S. Lee). This study used the stratification variables and sampling weights designated by the Korean Centers for Disease Control and Prevention, which were based on the sample design of each survey year. Sampling weights were adjusted for non-response according to demographic factors after the surveys were completed. The SAS Survey procedure was performed with cluster as a sampling-district variable. The SAS Surveyreg procedure was used to compare mean differences between groups, and the SAS Surveyfreq procedure was used to compare proportion differences between groups. Values are presented as mean ± SE for continuous variables, or as the number (%) of subjects for categorical variables. Descriptive statistics are presented for the four groups based on sleep duration and then analyzed using ANOVA or Chi-square test for distributed variables. Subgroup analysis was performed using Turkey's multiple comparison. Sleep duration associated with body size phenotype was calculated using ANCOVA after controlling for potential covariates of age, sex, smoking status, alcohol consumption, physical activity, income, and education. Data were analyzed using SAS for Windows (version 9.3, SAS Institute, Cary, NC, USA). A P value <0.05 indicated statistical significance.

3. Results

Baseline characteristics of the study population are shown in accordance with sleep duration in Table 1. According to sleep duration, there were apparent differences in age, gender, BMI, waist circumference, systolic and diastolic blood pressure among the study groups (all P <0.05).

Table 2 summarizes the prevalence of metabolic disorders in the four body phenotypes. According to body size phenotype, the prevalence of components of metabolic syndrome was significantly different (P <0.001). Fig. 1 shows that the obese population (MHO, MAO) had a higher proportion of individuals who slept <6 h/d, whereas the non-obese population (MHNW, MANW) slept longer than the obese population.

Table 3 shows the comparisons of the mean sleep duration among the four groups. Sleep duration was significantly different according to body size phenotype after adjusting for confounding factors, such as age, gender, smoking status, alcohol consumption, physical activity, income, and education. We further examined the differences in sleep duration according to obesity or metabolic health status. Unadjusted analysis demonstrated that obese group had a significantly shorter sleep duration compared to the non-obese group (6.82 ± 0.03 vs. 7.00 ± 0.02, P <0.001). Metabolically unhealthy subjects also showed shorter sleep duration compared to metabolically healthy subjects (6.84 ± 0.03 vs. 6.98 ± 0.02, P <0.001) (Supplementary Table 1). However, multivariate analysis revealed that obese people are shorter sleepers than the non-obese people (18 < BMI < 25 kg/m^2; 6.78 ± 0.04 vs. BMI ≥25 kg/m^2; 6.93 ± 0.03, P <0.001), but the difference in sleep duration between the metabolically unhealthy and healthy group was attenuated (6.85 ± 0.04 vs. 6.90 ± 0.03, P = 0.168).

4. Discussion

Although obesity is regarded as the main cause of various metabolic disorders, recent studies have suggested that a unique subset of obese individuals may exist without metabolic derangements, known as MHO. Epidemiological and clinical studies reported that the prevalence of MHO subjects may vary between 10% and 40% among general population [16]. In the present study, 1278 individuals among 9077 subjects (14.1%) were classified as being in the MHO group. On the contrary, the MANW individuals, despite having a normal BMI, displayed harmful metabolic profiles, such as high blood pressure and glucose levels, insulin resistance, and atherogenic lipid parameters. In light of the world-wide epidemic of obesity, the current "one size fits all" approach for the management of obesity may not be appropriate [17]. At the present time, there are only limited data regarding the determining factors of the MHO or

Table 1 – Baseline characteristics of the study population.

Sleep duration (h/d)					
	≤6 (n = 3325)	7 (n = 2815)	8 (n = 2212)	≥9 (n = 725)	P *
Age (years)	42.34 ± 0.31	40.53 ± 0.32	40.11 ± 0.37	39.89 ± 0.72	<0.001
Male	60.4% (0.96)	56.6% (1.04)	53.7% (1.21)	52.0% (2.04)	<0.001
BMI (kg/m^2)	24.04 ± 0.07	23.70 ± 0.06	23.33 ± 0.07	23.09 ± 0.16	<0.001
WC (cm)	81.96 ± 0.22	80.94 ± 0.21	80.11 ± 0.24	79.59 ± 0.43	<0.001
FPG (mmol/L)	5.34 ± 0.03	5.29 ± 0.02	5.26 ± 0.03	5.28 ± 0.05	0.175
SBP (mmHg)	115.64 ± 0.34	114.27 ± 0.36	113.96 ± 0.41	113.47 ± 0.68	0.003
DBP (mmHg)	76.29 ± 0.25	75.29 ± 0.28	74.90 ± 0.29	73.44 ± 0.48	<0.001
TG (mmol/L)	1.56 ± 0.03	1.51 ± 0.03	1.50 ± 0.03	1.53 ± 0.05	0.615
HDL-C (mmol/L)	1.32 ± 0.01	1.32 ± 0.01	1.32 ± 0.01	1.34 ± 0.02	0.631
HbA1c (%)	7.56 ± 0.18	7.20 ± 0.14	7.42 ± 0.17	7.83 ± 0.28	0.164
Insulin (μIU/mL)	9.84 ± 0.12	10.27 ± 0.16	9.95 ± 0.14	10.18 ± 0.20	0.116
Alcohol	67.1% (1.04)	66.3% (1.04)	64.6% (1.18)	62.0% (2.23)	0.095
Smoking	32.4% (0.98)	29.7% (1.06)	31.2% (1.19)	32.9% (2.21)	0.271
Physical activity	11.6% (0.70)	13.6% (0.80)	12.0% (0.92)	10.4% (1.38)	0.132
Hypertension	19.1% (0.77)	15.3% (0.75)	14.4% (0.92)	15.0% (1.44)	<0.001
Diabetes	6.1% (0.44)	5.8% (0.48)	6.2% (0.52)	6.2% (1.00)	0.940

BMI, body mass index; WC, waist circumference; FPG, fasting plasma glucose; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglycerides; HDL-C; high density lipoprotein cholesterol.
* P-value by ANOVA or Chi-square test as appropriate.

MANW phenotype. Differences in subclinical inflammation, ectopic fat accumulation, cell size, and gene-expression profiles in adipocytes have been suggested to be associated with the development of the different body size phenotypes, such as MHO or MANW [16]. Velho et al. demonstrated that physical activity was positively associated with MHO [18]. In contrast, Appleton et al. reported that MHO was a transient state to metabolic disorders and was associated with smoking, socioeconomic advantage, and physical inactivity [19]. In this study, we first found that sleep duration is significantly different according to body size phenotype in a Korean population, even after adjusting for various kinds of lifestyle and socio-economic factors.

Previous studies have provided evidence that demonstrates a relationship between sleep duration and components of metabolic syndrome. Experimental reduction of sleep duration has a deleterious effect on glucose metabolism [20]. Both cross-sectional and prospective epidemiologic studies have provided consistent evidence of the relationship between short sleep duration and the prevalence or incidence of type 2 diabetes [20]. This may be associated with dysregulation of the hypothalamus–pituitary–adrenal axis and insulin resistance [21]. In the Nurses Health Study, both longer than average and shorter than average sleep durations were associated with the incidence of diabetes in women [22]. The relative risk for subjects with short sleep duration (≤5 h per day) was 1.57 (95% CI = 1.28–1.92) and those with long sleep duration (≥9 h per day) was 1.47 (95% CI = 1.19–1.80), respectively [22]. Gottlieb et al. also reported that a sleep duration of 6 h or less or 9 h or more was associated with increased prevalence of impaired glucose tolerance (IGT) and diabetes [4]. Furthermore, sleep duration has been suggested to have an important role in the development of hypertension. A recent meta-analysis demonstrated that a short sleep duration was associated with an increased

Table 2 – The prevalence of components of metabolic syndrome according to body size phenotype.

	MHNW			MANW			MHO			MAO			P *
Abdominal obesity	453	7.1%	(0.38)	603	42.5%	(1.67)	690	50.2%	(1.70)	1037	83.3%	(1.38)	<0.001
High TG	657	13.0%	(0.53)	850	66.3%	(1.58)	193	15.8%	(1.26)	914	77.1%	(1.36)	<0.001
Low HDL cholesterol	1880	36.8%	(0.93)	1003	76.6%	(1.49)	571	46.4%	(1.70)	1033	85.7%	(1.03)	<0.001
Elevated blood pressure	730	13.3%	(0.64)	799	65.3%	(1.70)	233	20.6%	(1.47)	706	61.6%	(1.67)	<0.001
Elevated blood glucose	506	8.9%	(0.46)	1011	76.8%	(1.33)	116	8.8%	(0.90)	620	48.9%	(1.70)	<0.001
Number of components of metabolic syndrome													
0	2155	42.6%	(0.83)	6	0.5%	(0.22)	143	12.0%	(1.09)	–			
1	1984	37.2%	(0.79)	57	4.8%	(0.73)	467	36.6%	(1.62)	–			
2	1121	20.2%	(0.61)	122	9.1%	(0.99)	668	51.4%	(1.69)	–			
3	–			667	51.5%	(1.72)	–			701	58.5%	(1.59)	
≥4	–			464	34.2%	(1.70)	–			522	41.5%	(1.59)	

MHNW, metabolically healthy and normal weight; MANW, metabolically abnormal but normal weight; MHO, metabolically healthy but obese; MAO, metabolically abnormal obese.
* P-value by ANOVA or Chi-square test as appropriate.

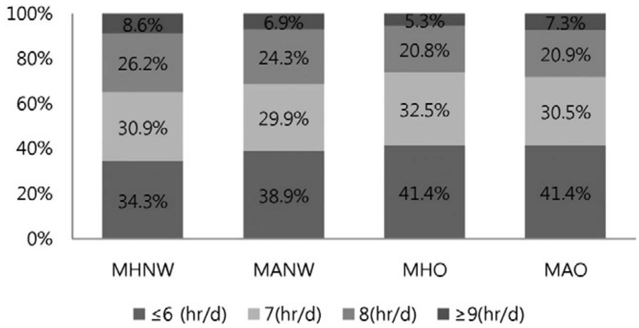

Fig. 1 – The proportion of sleep duration among the four body size phenotypes.MHNW, metabolically healthy and normal weight; MANW, metabolically abnormal but normal weight; MHO, metabolically healthy but obese; MAO, metabolically abnormal obese.

risk of prevalent hypertension (OR = 1.20, 95% CI = 1.09–1.32, P <0.001) and an increased risk of incident hypertension (RR = 1.33, 95% CI = 1.11–1.61, P = 0.002) [7]. Moreover, there was a significant relationship between long sleep duration and the risk of prevalent hypertension (OR = 1.11, 95% CI = 1.05–1.17, P <0.001), confirming the presence of a U-shaped association [7]. As a mechanism for the association, short sleep duration could elevate the activity of the sympathetic nervous system and amplify physical and psychosocial stress, which can increase the average 24 h blood pressure and heart rate [23]. In addition, elevated cortisol and pro-inflammatory cytokine levels, endothelial dysfunction, and renal impairment could also contribute to hypertension [24]. However, the association between long sleep duration and obesity-related cardiometabolic disorders may be explained by some other residual confounding factors related with long sleep duration, such as unemployment status, low education, and socio-economic status [25]. The present study showed differences in components of metabolic syndrome, such as hyperglycemia and hypertension, according to body size phenotype.

In addition, a prospective cohort study of 496 young adults showed a negative relationship between sleep duration and BMI [3]. Another prospective study that included 1001 participants found that overweight and obese subjects slept less than those with a normal BMI and this trend was reversed in the extremely obese patients [26]. The association between sleep duration and obesity was confirmed in the current study using data from a representative sample of Korean men and women. This relationship persisted even after adjusting for various kinds of confounding factors, such as age, gender, smoking status, alcohol consumption, physical activity, income, and education. Interestingly, although the metabolically unhealthy group also showed shorter sleep durations compared to the metabolically healthy group in an unadjusted model, this association was attenuated after adjusting for confounding factors. These results may suggest that obesity itself is more independently associated with sleep duration than metabolic deterioration in a Korean population. Further studies to explore the mechanistic role of sleep duration on obesity and metabolic disorders may be needed.

Although the mechanism linking sleep duration and obesity has not been clearly defined, short sleep duration deteriorates multiple metabolic pathways, leading to increased appetite, possibly reduced energy expenditure, as well as hormonal and immunological changes [27]. Individuals with short sleep duration tend to feel more fatigue and have decreased daytime physical activity [2]. Gupta et al. reported that the OR of obesity increased by 80% for each hour of decrease in sleep duration and daytime physical activity diminished by 3% for each hour of increase in sleep disturbance [28]. In addition, a longer awake time may provide more opportunity for food intake [29]. Sleep restriction is associated with decreased secretion of the anorexigenic hormone leptin and increased secretion of the orexigenic hormone ghrelin, which result in augmented hunger and food intake [30,31]. Furthermore, sleep restriction has been reported to increase pro-inflammatory cytokines, such as tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) [32]. Chronic low-grade elevation of pro-inflammatory cytokines is known as subclinical inflammation, which is frequently found in obesity. In addition, partial and total sleep deprivation resulted in elevated plasma and salivary cortisol, a hormone which is closely associated with obesity and metabolic syndrome [33,34]. Obesity and insulin resistance can result from these hormonal changes associated with sleep duration [27].

Several limitations of our study should be noted. First, as with most of the previous studies, the information on sleep duration was self-reported. However, the data in the present study were obtained by trained investigators using the detailed questionnaire in the National survey performed by the Korean government. Moreover, self-reported sleep duration has been validated in previous studies using quantitative sleep assessments [25]. Second, the cross-sectional design of the study does not allow for establishing the causality of an

Table 3 – Mean sleep duration among the four body size phenotypes.

	MHNW	MANW	MHO	MAO	P^*
Unadjusted	7.02 ± 0.02	6.88^{a} ± 0.04	6.80^{a} ± 0.04	6.83^{a} ± 0.04	<0.001
Model 1	6.97^{a} ± 0.02	6.96^{ab} ± 0.04	6.77^{c} ± 0.04	6.84^{bc} ± 0.04	<0.001
Model 2	6.94^{a} ± 0.04	6.91^{a} ± 0.05	6.73^{b} ± 0.05	6.82^{ab} ± 0.05	<0.001

Model 1 adjusted for age and gender.
Model 2 adjusted for Model 1 plus smoking status, alcohol consumption, physical activity, income and education.
^{a,b,c} Same letters indicate no statistical significance based on Tukey's multiple comparison.
MHNW, metabolically healthy and normal weight; MANW, metabolically abnormal but normal weight; MHO, metabolically healthy but obese; MAO, metabolically abnormal obese.
* P-value by ANOVA or ANCOVA as appropriate.

observed association. However, the present study does have some strengths. This study was performed using KNHANES data, as it ensured reliable countrywide sampling and utilized a survey that was both large-scale and nationally representative. Furthermore, the current study analyzed the association between body size phenotype and sleep duration after adjustment for various potential confounding factors, using proper statistical analyses considering the stratified multistage sampling design.

In conclusion, we found that sleep duration is independently associated with body size phenotype, such as MANW or MHO, after adjusting for confounding factors in the present study using the data from a representative sample of Korean men and women. Furthermore, obesity may be more important factor that explains this association than metabolic abnormalities in Korea adults.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.metabol.2014.12.001.

Authors' contributions

K.M. Choi designed this study. J.Y. Ryu and K.M. Choi wrote the manuscript and performed the research. J.S. Lee analyzed the data set. H.C. Hong, H.Y. Choi, H.J. Yoo, J.A. Seo, S.G. Kim, and N.H. Kim collected data and offered technical support. S.H. Baik, and D.S. Choi reviewed and edited the manuscript.

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Conflicts of interest

None.

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